	Rhodamin.	Pyronin.	Auramin.
Micrococcus aureus (1)	0	+++	+++
Streptococcus (92)	+++	+++	+++
Micrococcus catarrhalis (6)	0	+++	+++
Bacillus anthracis (14)	0 to ±	+++	+++
Actinomyces (15)	0 to ±	+++	+++

SUMMARY. 1. The inhibitory effect which certain violet dyes, like gentian violet, methyl violet, dahlia, etc., exercise upon certain bacteria is not referable to their violet color, but to their chemical structure as triamino triphenyl methanes, and is intimately dependent upon their basic auxochromic groups.

2. Acid dyes irrespective of their chemical structure and color, do not possess this inhibitory power.

TYPHLO-ALBUMINURIA.[1]

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IT is not rare to find a clinician assigning an alimentary origin to a case of albuminuria when it cannot be attributed to an existing kidney disease and when the albuminuric phenomenon follows the ingestion of generous amounts of meat, cheese, milk, gelatin, or raw egg albumen. While it is true that in persons with faultless kidneys the ingestion of the raw whites of three or four eggs may result in the excretion of the unchanged ovialbumin, very little, indeed, is known of the albuminurias supposedly occurring in the wake of an overplus of the other protein foodstuffs. In such albuminurias only serins—serum albumin together with serum globulin or serum albumin alone—and no blood-foreign protein are excreted by the urine.

It is for this reason that, apart from ovialbuminuria occasionally following the intake of excessive amounts of raw egg albumen, no definite connection between protein ingestion and the urinary albumin has as yet been established. Every food protein is altered prior to absorption, and though its end-products will ultimately reach the renal secretion, it will not therein reappear in its native physicochemical formation.

There is, moreover, no probability that such a relationship can ever be positively demonstrated so long as we hold to the belief that the protein substance designated by us as serum albumin must have formed a constituent part of the blood. This assumption not only presupposes that the living organism exhibits its contents of serum albumin exclusively in the blood plasma, but also that the escape to this protein from the body must ensue by way of the kidneys.

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[1] Read at the Annual Meeting of the Medical Society of the State of New York at Rochester, April 30, 1913.

The albumin of the lymph, however, is entirely identical with serum albumin. Inasmuch as we cannot differentiate one from the other, we would be unable to recognize the source of albumin appearing in the urine were it not for the facts that globinuria is a conspicuous concomitant of serum albuminuria while it is decidedly less manifest or entirely absent in lymphuria, and that the latter usually shows considerable numbers of lymphocytes on subjecting a sediment to microscopic examination.

If we accept the fact that albumin may be transmitted to the urinary fluid by traversing the lymph channels—and I have furnished sufficient evidence that this may be the case—[2] then it is even possible that the well-developed network of renal lymphatics need not play any role whatever in this process, for the lymph albumin may enter the urinary tract by way of the lymph-paths leading to the ureters and the bladder. The superficial ureteral and vesical lymph vessels directly adjoin the upper epithelial layer of the mucosa communicating with it, and thus a portion of their lymph content may under certain conditions exude into the lumen of the ureter and the interior of the bladder.

In alimentary albuminuria in general and typhlo-albuminuria in particular there is one main channel, and in uncomplicated cases it is probably the only one, through which the protein is conveyed to the urine—the lymphatics. It is the route taken by the chyle in the condition known as chyluria, and a similar if not the same one often followed by the colon bacillus invading the urinary tract when the infection is not of the ascending type. Nevertheless, I am not as yet prepared to positively state whether or not alimentary albuminuria is a true lymphuria, for one of the characteristic features of this, the presence of large numbers of lymphocytes, is generally wanting. At the same time there ensues no or but an inconsiderable globulin excretion in the albuminurias of intestinal origin. This latter factor at once precludes the blood as the protein carrier in these types of albuminuria.

It might be possible that the criteria of lymphuria as advanced by me are too stringent, and that this condition may exist even when the urine does not show considerable numbers of lymphocytes that are not the result or the accompaniment of destructive processes. Assuming, then, that this be the case, an alimentary albuminuria would be a lymphuria—an assumption which is so much more plausible than that which tries to trace the urinary albumin back to the blood current.

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[2] Heinrich Stern, Lymphuria and its Clinical Status (Arch. Diag., April, 1913), The Clinical Evidence of Lymphuria (Med. Rec., May 31, 1913), and Ueber Lymphurie und ihren klinischen Status (Berl. klin. Woch., October 13, 1913).

Typhlo-albuminuria is an alimentary albuminuria proceeding from the cecum. It is probably the most frequent of all the intestinal albuminurias. It has a recidivating character, that is, it disappears and returns at irregular intervals, and seems to ensue quite independently from the protein intake just preceding it. It may be continuous for several days and again be absent for weeks at a time. Typhlo-albuminuria invariably occurs in persons with habitual constipation, which latter appears to stand in more or less etiological connection with it. Its immediate causative factors are probably analogous to those permitting the transmigration of the colon bacillus from the cecum to the renal pelvis, ureter, or bladder and terminating in colipyelocystitis. These pathogenic factors are cecal atony, the absorption of nearly or all the liquid from the cecum, the resulting extreme dryness of the feces, and coprostasis. At any rate, we cannot speak of a typhlo-albuminuria unless the cecum appears to be at the bottom of the albuminuric phenomenon. The latter may supervene during or after a paroxysm of severe griping pain in the cecum (oftentimes a characteristic of cecal atony), but it is more frequent in those instances of typhlatonia in which there is no definite paroxysm of pain, but constant discomfort due to fulness, compression, or traction at the site of the cecum. While it is now a comparatively easy matter to recognize typhlatonia and to differentiate it from chronic appendicitis[3] the appendix itself may be adherent to the cecum and thus occasion or aggravate the latter's atonic state.

The structurally or functionally defective cecum is bound to more of less disturb the intra-abdominal equilibrium in general and the visceral lymphatics in particular. The latter, including those of the cecum itself and its mesocolon, become readily overfilled and a lymph stasis may ensue which in itself may give rise to abdominal and lumbar discomfort. The escape of some of the contents of the engorged lymph channels through newly formed lymph varices near the surface of the lining epithelium of the urinary tract is but a natural consequence. Whenever an anastomosis between the cecal or mesenteric lymphatics on the one hand, and those of the urinary apparatus on the other, is effected, the albuminuric phenomenon may even ensue in the absence of any considerable cecal disturbance, and the typhlo-albuminuria may thus attain a certain degree of chronicity.

The occurrence of typhlo-albuminuria does not seem to depend so much upon the amount of protein ingested at any one time as on the interrupted draining of the lymph stream into the venous system. There is set up a localized lymph stasis resulting in the absolute augmentation of lymph albumin. While no albumin can be detected in the urine immediately before the lymph paths communicate with any of the lumina of the urinary apparatus, it will be found by the ordinary clinical methods as soon as the anastomosis has become patent. It is this sudden appearance of urinary albumin which has given cause to the unjustified assumption (excepting the circumstance of the ovialbumin) contending that this type of albuminuria is the direct consequence of the increased ingestion of protein material.

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[3] Heinrich Stern, Atony of the Cecum: A Procedure Facilitating its Recognition, Arch. Diag., October, 1912, and Atony of the Cecum (Typhlatonia), Arch. Diag., January, 1913).

Besides an albuminuria, a visceral lymph stasis may engender other clinical phenomena. There exists, for instance, an albumino-glycosuria which is of lymphatic origin, and which I shall describe in detail as soon as I have collected all my necessary data.

In 2 of my 4 cases of true lymphuria[4] I have encountered traces of fatty material on the surface of the urine upon standing for some time. I have not as yet observed any urinary fat excretion in cases of typhlo-albuminuria or alimentary albuminuria in general, but it must be known that my investigation of these particular types of albuminuria were not begun before May, 1912, and that the material at my disposal was limited to 10 cases, namely, 5 of typhlo-albuminuria and 5 in which the direct cause of the albumin excretion was evidently located in another segment of the colon; 2 of the latter cases were associated with a glycosuria.

Theoretically reasoning, typhlo-albuminuria ought to be a unilateral, that is, a right-sided albuminuria. There is not only the close proximity of the cecum to the right half of the urinary apparatus and the bladder (I recall the substitution of the cecum for the resected bladder by P. Lengemann,[5] but also the intimate lymphatic communication between these organs. To my regret in but one of my cases was I permitted to obtain the separate urine from each kidney by ureteral catheterization. In this case, which is reported in the following, the albuminuria proved to be bilateral, though its intensity degree was from three to four times larger in the secretion from the right than in that derived from the left ureter.

Case.—Male, aged thirty-two years; well-developed. No history of scarlatina or diphtheria. Gonorrheal infection ten years before. Six years before was accepted by a life insurance company, but the previous year was rejected by the same and two other insurance companies on account of albumin in his urine. Has since been under treatment by his family physician, without avail, however, as far as the albuminuria is concerned. This was irregularly intermittent: it was present for a few days in succession at all hours of the day; at other times it would be found between 3 o'clock in the afternoon and bedtime; and again at other times it would not appear for some days. Complains of fulness and dragging in the right side of the abdomen, occasional sharp pains, lasting for from half an hour to an hour and a half, in the right inguinal region; constipation and hemorrhoids. There was no heart or appreciable bloodvessel disease; the pressure over both radial arteries was 140 mm. Hg. The neurological findings were negative. The twenty-four hours' urinary output fluctuated between 1400 and 1800 c.c. The specific gravity varied between 1023 and 1026. The urine was invariably of a normal acid degree. The albuminous urine never contained globulin. The epithelia distinguished came mostly from the upper layers of the ureters, bladder, and urethra. Besides there were some prostatic epithelia. The few leukocytes encountered were well granulated and of good constitution. In addition there was a limited number of small-calibered calcium oxalate crystals. No renal formed elements or tube-casts were found in the numerous urine specimens submitted to microscopic examination. The separate urines obtained by ureteral catheterization evidenced bilateral albumin excretion; however, the urine from the right ureter contained three or four times as much protein as the one obtained from the left ureter. The test for the functional activity of the kidneys with phloridzin, of which 1 cm. (0.01) was injected hypodermically, showed the excellent working powers of these organs, as their secreting epithelium produces not less than 8.63 grams glucose, that is, about six times more than is furnished by kidneys yet considered physiologically efficient.

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[4] Arch. Diag., April, 1912.

[5] Zentralbl. f. Chir., 1912, No. 50.

The abdominal organs appeared normal, with the exception of the ascending colon and the cecum, which were both distended, the former by gases and the latter by liquid, and apparently also by solid material. There was considerable pressure sensitiveness over the cecum, which was readily mapped out and demarcated from the ascending colon by percussion. Arising from within it there were distinct gurgling sounds. Stern's procedure confirmed the diagnosis of cecal atony.

The albuminuria in this case was entirely independent from the protein intake. It was even more intense on the days the patient reduced the total food intake and abstained entirely from animal proteins. The enhanced intensity degree of the albuminuria continued sometimes on the following day.

A mixed diet rich in cellulose, the imbibing of not less than two liters of water per day, regulated bodily exercise, and the wearing of a cecal support afforded cecal relief, and also lessened the albuminuria days and the absolute output of albumin.

Our knowledge about alimentary albuminuria is very meager, and the few conceptions we entertain concerning it are in need of a thorough revision. An alimentary albuminuria in the first instance should not so much signify the excretion of ingested albumin as the egestion of proteid material due to some anomaly of the alimentary tract. Typhlo-albuminuria is a true alimentary albuminuria in which the sequence of cause and effect can clinically be demonstrated with certainty in well-nigh every instance.